Lead in Soil: Issues and Guidelines

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Soil-dust Lead and Childhood Lead Exposure as a Function of City Size and Community Traffic Flow: The Case for Lead Abatement in Minnesota

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Abstract
This paper reports the results of a survey conducted by the State of Minnesota after the January 1, 1986 leaded petrol phasedown. The survey collected data on the soil-dust lead and children's blood lead concentrations in the oldest census tracts of Minneapolis, St Paul, Duluth, St Cloud and Rochester, Minnesota. The results reveal fundamental differences among cities. The proportion of children with blood lead concentrations equal or greater than 25 μg/dL was 3.7% in Minneapolis, 3.1% in St Paul, and 0.7% in Duluth. The proportion of children with blood lead concentrations equal or greater than 15 μg/dL was 17.7% in Minneapolis, 14.9% in St Paul, and 9.8% in Duluth. No children in the small cities of St Cloud or Rochester had blood lead contents above 15 μg/dL. Soil lead concentrations of yards, streetsides and foundations showed a similar trend with the highest being in Minneapolis and the lowest in Rochester. For example, midyard soils above 150 mg/kg range from 67.5% in Minneapolis, 51.0% in St Paul, and 46.2% in Duluth, to 4.8% in St Cloud, and 4.5% in Rochester. Although the lead contents of foundation soils were highest, the concentrations found were not simply related to age of community dwellings. Children's blood lead and environmental lead contents were directly related to city size, which in turn is related to traffic density as well as quantity of

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community traffic flow. The survey provides essential information about the groups at risk, major sources of lead, and the geography of lead to set priorities for abatement and primary prevention of lead exposure in Minnesota.

Introduction

Exposure to children of lead–contaminated soil and dust is the subject of this paper. The idea that high soil-dust lead concentrations will be found in the inner-city communities of all major cities was a major conclusion from an empirical study of garden soils conducted in metropolitan Baltimore (Mielke *et al.*, 1983). As a result, children living in these communities have a higher probability of being more exposed to lead than children living in less contaminated urban locations. In the Baltimore study the probability of explaining the degree of clustering of high lead soils toward the centre of the metropolitan area by chance alone was about one in 10^{-23}. A follow up study in Minneapolis of the general urban pattern and specific residential community soil lead concentrations further supported the idea that clustering of high lead soils has occurred within all major urban centres and that this phenomenon is not unique to Baltimore (Mielke *et al.*, 1984). In addition, soil lead transects of four Minnesota cities, Minneapolis, St Paul, Duluth and Rochester revealed that city size and geographic factors played a major role in the amount of lead that has accumulated within a specific urban community (Mielke *et al.*, 1984/85).

A major question that could not be answered previously in Minnesota was the degree of lead exposure to childhood populations living in the various city environments. As a result of a law passed by the Minnesota legislature in 1985, new data have been collected that makes it possible to examine the relationships between soil lead, city size, selected community features, and childhood lead exposure. It is the purpose of this paper to evaluate the quantitative relationship between soil lead and childhood lead exposure as empirically observed among cities and within urban communities of Minnesota. None of the communities are sites of lead mines or lead smelting facilities. These cities represent a cross section of light industrial communities and are similar to many cities in the United States. This analysis has a direct bearing on public policy formulation concerning amelioration of the lead exposure problem in nonbindustrial communities of Minnesota and elsewhere.

Methods

The data for this paper were collected by the Minnesota Pollution Control Agency (MPCA) and the Minnesota Department of Health (MDH) as required by the Minnesota Legislature, 1985 Laws, First Special Sessions, Chapter 14, Article 19, Section 11. The data were collected to describe ". . . the extent of lead contamination in the soil, the lead concentrations in the blood of populations at contaminated sites, the size of the population at risk from exposure to lead in the soil . . .". State agency staff collected

two different data sets. One data set consists of 187 blood lead cases in Minneapolis, matched and paired with maximum soil lead contents of residential sites for each case. The second data set consists of 1,266 unpaired blood lead cases and 1,337 unpaired soil lead cases collected from several communities in Minnesota. We focused our analysis on the large unpaired data set.

Soil or dust samples were collected according to an MPCA protocol. Five cities (Minneapolis, St Paul, Duluth, St Cloud and Rochester) were sampled (see Figure 1). Census tracts in these five cities were identified and ranked according to: 1) childhood population under the age of 5; 2) black population; 3) residences with an income of less than $15,000/year;

Figure 1. *The following cities (with 1980 census population data in parentheses) were the sites of the Minnesota soil lead and blood lead survey: Minneapolis (371,000), St Paul (271,000), Duluth (93,000), St Cloud (43,000) and Rochester (58,000).*

4) residences with an income of less than $7,500/year; 5) number of residences built prior to 1960. Within selected census tracts, soils were collected from around foundations, in the middle of yards, and near streets. All soils were sampled from the top 2 cm.

From January 1, 1986 there was a 91% reduction of lead content in petrol. Blood lead samples were all collected at least seven months after this date. The age of the children tested was nine months to six years old. The first group of children was tested in late summer and early fall of 1986 and included children living in St Paul and South Minneapolis. The remainder of the blood lead sampling was done in the summer of 1987 and included children in St Paul, North Minneapolis, Duluth, St Cloud and Rochester. The blood lead surveys were generally done in response to the finding of 1,000 mg Pb/kg concentration in the soil. Following an MDH protocol, blood lead testing was conducted on a sample of children living up to five blocks away from the 1,000 mg Pb/kg sample. Census tracts that had even a single soil containing 1,000 mg Pb/kg or more were thereby subject to response, but blood lead surveys were also conducted in census tracts where soil or dust samples were not collected. This approach produced a relatively representative group of blood leads within the collection sites of the state. Both soil and blood samples were generally collected in the oldest census tracts of the state of Minnesota.

We analyzed the blood lead and soil lead data by first stratifying the data by city and, secondly, in the Twin Cities, by stratifying communities

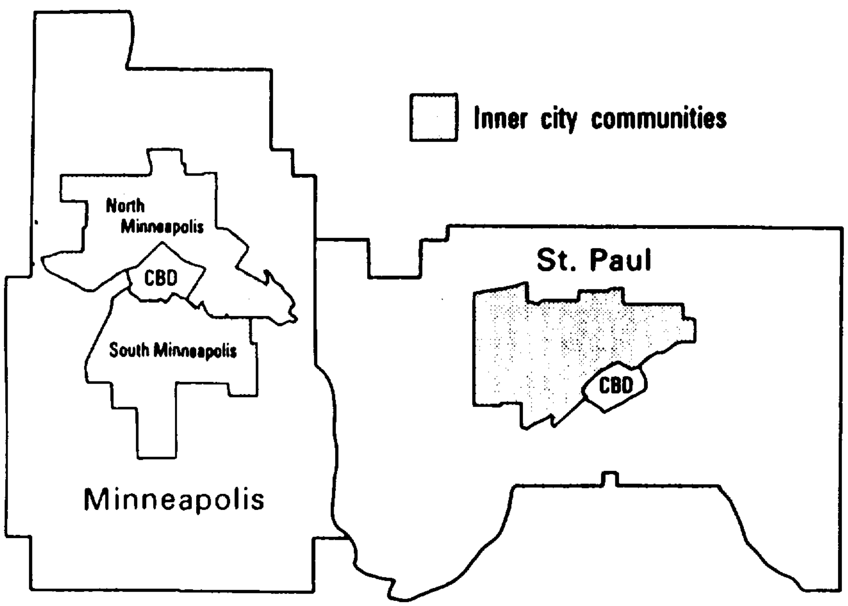

Figure 2. *Map of the stratification of census tracts in the Twin Cities according to inner- and outer-city location and by North and South inner-city communities in Minneapolis (CBD=central business district).*

according to inner and outer city location. In Minneapolis, stratification was also done between two distinct areas of the inner city, the communities of North and South Minneapolis. The stratification of inner and outer city communities derives from an urban geography map of the Twin Cities published by Martin and Lanegran (1982). Figure 2 shows the more refined stratification of the Twin Cities metropolitan area by outer city and inner city of Minneapolis and St Paul as well as inner city North and South Minneapolis. In all cases we focused on statistical descriptions of various stratified groups of soils and various stratified populations of children. The stratified data sets were analyzed by using one sided Fisher's exact test to determine the differences and statistical significances between communities.

Results

The frequencies and analytical results of various soil lead values and population blood lead concentrations are shown by city subset in Tables 1–8. Tables 1–3 show the frequencies of soil lead concentrations for yards, streetbsides, and foundations, respectively, for each city. The same tables also present the pairwise p-values for the one sided Fisher's exact test for comparisons between soil lead values for each of the cities in relationship to each of the other cities. Tables 5–7 show the frequencies of soil lead for yards, streetsides and foundations, respectively, for Twin Cities communities, along with the one sided Fisher's exact test results. Tables 4 and 8 show the various blood lead concentrations exhibited by children, as well as the pairwise P-values for the one sided Fisher's exact test of blood lead concentrations for each of the cities and Twin Cities communities in comparison with each other. From Tables 1–4 it is shown that soil lead concentrations in cities and the blood lead concentrations of the childhood population generally vary in a lock-step manner with each other. The P-

Table 1. *Frequency table of yard soil lead data for Minnesota cities.*

Soil lead (mg/kg)	Minneapolis	St Paul	Duluth	St Cloud	Rochester
	(percent of samples)				
≤50	6.4	20.5	13.5	76.2	68.2
51–150	26.0	28.5	40.4	19.0	27.3
151–300	36.2	30.0	19.2	4.8	0.0
301–600	21.9	13.5	21.2	0.0	0.0
601–1,200	8.7	5.0	3.8	0.0	4.5
≥1,201	0.8	2.5	1.9	0.0	0.0
Total	100.0	100.0	100.0	100.0	100.0
(N)	(265)	(200)	(52)	(21)	(22)

One-sided Fisher's exact test *P*-values for ≤150 *versus* ≥151 comparisons.

	St Paul	Duluth	St Cloud	Rochester
Minneapolis	$0.22\ 10^{3}$	$0.31\ 10^{2}$	$0.10\ 10^{7}$	$0.40\ 10^{6}$
St Paul		0.32	$0.18\ 10^{4}$	$0.98\ 10^{5}$
Duluth			$0.41\ 10^{3}$	$0.28\ 10^{3}$
St Cloud				0.74

Table 2. *Frequency table of street-side soil lead data for Minnesota cities.*

Soil lead (mg/kg)	Minneapolis	St Paul	Duluth	St Cloud	Rochester
	(percent of samples)				
≤50	5.4	16.0	12.2	40.8	40.8
51–150	38.1	40.8	43.9	37.0	48.1
151-300	31.5	34.9	29.3	14.8	11.1
301–600	19.6	8.3	12.2	7.4	0.0
601–1,200	4.8	0.0	2.4	0.0	0.0
≥1,201	0.6	0.0	0.0	0.0	0.0
Total	100.0	100.0	100.0	100.0	100.0
(N)	(168)	(169)	(41)	(27)	(27)

One-sided Fisher's exact test *P*-values for ≤150 *versus* ≥151 comparisons.

	St Paul	Duluth	St Cloud	Rochester
Minneapolis	$0.95\ 10^{-2}$	0.10	$0.80\ 10^{-3}$	$0.61\ 10^{-5}$
St Paul		0.54	$0.29\ 10^{-1}$	$0.82\ 10^{-3}$
Duluth			$0.57\ 10^{-1}$	$0.36\ 10^{-2}$
St Cloud				0.23

Table 3. *Frequency table of foundation soil lead data for Minnesota cities.*

Soil lead (mg/kg)	Minneapolis	St Paul	Duluth	St Cloud	Rochester
	(percent of samples)				
≤50	1.7	9.1	9.7	46.1	50.0
51–150	12.5	9.1	12.9	23.1	11.1
151–300	9.2	11.1	12.9	0.0	11.1
301–600	19.0	22.2	12.9	15.4	11.1
601–1,200	5.0	22.2	16.1	0.0	0.0
≥1,201	32.6	26.3	35.5	15.4	16.7
Total	100.0	100.0	100.0	100.0	100.0
(N)	(184)	(99)	(31)	(13)	(18)

One-sided Fisher's exact test *P*-values for ≤150 *versus*≥151 comparisons.

	St Paul	Duluth	St Cloud	Rochester
Minneapolis	0.23	0.17	$0.29\ 10^{-4}$	$0.24\ 10^{-4}$
St Paul		0.38	$0.31\ 10^{-3}$	$0.38\ 10^{-3}$
Duluth			$0.50\ 10^{-2}$	$0.86\ 10^{-2}$
St Cloud				0.47

value between both soil lead and blood lead concentrations becomes more extreme as the difference in city size becomes greater: note the similarities between St Paul and Duluth and between St Cloud and Rochester (see Figure 3).

From Tables 1–8 it can be seen that the highest population blood lead concentrations and environmental soil lead concentrations are found in the community of inner city South Minneapolis. The next highest blood lead and soil lead concentrations are found in the inner city of St Paul. A group

Table 4. *Frequency table of blood lead data for Minnesota cities (1986 and 1987 data).*

Blood lead (μg/dL)	Minneapolis	St Paul	Duluth	St Cloud	Rochester
	(percent of samples)				
0–4	28.2	37.4	55.2	42.2	98.1
5–9	32.7	32.7	21.0	46.7	1.9
10–14	21.4	14.9	14.0	11.1	0.0
15–19	8.8	6.4	7.7	0.0	0.0
20–24	5.3	5.5	1.4	0.0	0.0
25–29	1.9	2.2	0.7	0.0	0.0
≥30	1.7	0.9	0.0	0.0	0.0
Total	100.0	100.0	100.0	100.0	100.0
(N)	(571)	(455)	(143)	(45)	(52)

One-sided Fisher's exact test *P*-values for ≤9 *versus*≥10 comparisons.

	St Paul	Duluth	St Cloud	Rochester
Minneapolis	$0.13\ 10^{-2}$	$0.36\ 10^{-3}$	$0.60\ 10^{-4}$	$0.27\ 10^{-10}$
St Paul		$0.94\ 10^{-1}$	$0.39\ 10^{-2}$	$0.31\ 10^{-7}$
Duluth			$0.48\ 10^{-1}$	$0.78\ 10^{-5}$
St Cloud				$0.18\ 10^{-1}$

Table 5. *Frequency table of yard soil lead data for Twin Cities communities.*

Soil lead (mg/kg)	Minneapolis inner-city North	South	Minneapolis Inner	Outer	St Paul Inner	Outer
			(percent of samples)			
≤50	1 11.5	0 9	4.5	10.2	8.3	25.7
51–150	26.2	17.2	20.4	37.5	26.7	29.3
151–300	36.1	39.7	38.4	31.8	33.3	28.6
301–600	19.7	28.4	25.4	14.8	21.7	10.0
601–1,200	6.5	13.8	11.3	3.4	5.0	5.0
≥1,201	0.0	0.0	0.0	2.3	5.0	1.4
Total	100.0	100.0	100.0	100.0		
(N)	(61)	(116)	(177)	(88)	(60)	(140)

One-sided Fisher's exact test *P*-values for ≤150 *versus* ≥151 comparisons

Place	P-value
North *versus* South Minneapolis inner-city	$0.41\ 10^{-2}$
Minneapolis inner-city *versus* outer-city	$0.18\ 10^{-1}$
St Paul inner-city *versus* outer-city	$0.71\ 10^{-12}$

of communities consisting of outer city Minneapolis, the community of North Minneapolis, outer city St Paul and Duluth exhibit similarities in exposure to lead and environmental lead concentrations. The small cities of St Cloud and Rochester have significantly lower soil lead concentrations, the children in these cities exhibit the lowest blood lead concentrations and have the greatest margin of safety from lead exposure of the five cities in the study.

Table 6. *Frequency table of street-side soil lead data for Twin Cities communities.*

Soil lead (mg/kg)	Minneapolis inner-city North	South	Minneapolis Inner	Outer	St Paul Inner	Outer
			(percent of samples)			
≤50	4.5	1.5	2.6	11.1	14.0	16.8
51–150	42.2	20.3	28.9	57.4	36.0	42.9
151–300	40.0	39.1	39.5	14.8	42.0	31.9
301–600	6.7	33.3	22.8	13.0	8.0	8.4
601–1,200	4.4	5.8	5.3	3.7	0.0	0.0
≥1201	2.2	0.0	0.9	0.0	0.0	0.0
Total	100.0	100.0	100.0	100.0		
(N)	(45)	(69)	(114)	(54)	(50)	(119)

One-sided Fisher's exact test *P*-values for ≤150 *versus* ≥151 comparisons.

Place	*P*-value
North *versus* South Minneapolis inner-city	$0.49\ 10^{-2}$
Minneapolis inner-city *versus* outer-city	$0.65\ 10^{-5}$
St Paul inner-city *versus* outer-city 0.16	

Table 7. *Frequency table of foundation soil lead data for Twin Cities communities.*

Soil Lead (mg/kg)	Minneapolis inner-city North	South	Minneapolis Inner	Outer	St Paul Inner	Outer
			(percent of samples)			
≤50	5.9	0.0	1.5	2.0	9.4	9.0
51–150	26.5	6.1	11.3	15.7	3.1	11.9
151–300	8.8	3.0	4.5	21.6	12.5	10.4
301–600	11.7	22.2	19.5	17.6	18.8	23.9
601–1,200	20.6	29.3	27.1	19.6	28.1	19.4
≥1,201	26.5	39.4	36.1	23.5	28.1	25.4
Total	100.0	100.0	100.0	100.0	100.0	100.0
(N)	(34)	(99)	(133)	(51)	(32)	(67)

One-sided Fisher's exact test *P*-values for ≤150 *versus* ≥151 comparisons.

Place	*P*-value
North *versus* South Minneapolis inner-city	$0.29\ 10^{-3}$
Minneapolis inner-city *versus* outer-city 0.27	
St Paul inner-city *versus* outer-city	0.23

Discussion

The state soil data support earlier findings that environments of the three largest cities of Minnesota are burdened with the highest concentrations of soil lead (Mielke *et al.*, 1984; Mielke *et al.*, 1984/1985). In the early studies it was noted that the non polluted background lead concentrations for surface soil samples of rural Minnesota were between 5 and 10 mg/kg (Mielke *et al.*, 1984); these low background concentrations are supported by other researchers who have reported that rural soils average 6.8 mg Pb/kg (SD 3.5 mg/kg, range 0.7b9.4) (Pierce *et al.*, 1982). In contrast with the

Table 8. *Frequency table of blood lead data for Twin Cities communities (1986 and 1987 data)* .

Blood lead (μg/dL)	Minneapolis inner-city North	South	Minneapolis Inner	Outer	St Paul Inner	Outer
			(percent of samples)			
0–4	35.2	23.9	28.4	27.9	27.4	44.5
5–9	38.6	29.4	33.0	32.2	30.5	34.3
10–14	18.6	17.4	17.9	27.4	18.4	12.5
15–19	4.8	12.4	9.4	7.7	9.0	4.5
20–24	1.4	10.5	6.9	2.4	8.4	3.4
25–29	1.4	4.1	3.0	0.0	4.2	0.8
≥30	0.0	2.3	1.4	2.4	2.1	0.0
Total	100.0	100.0	100.0	100.0	100.0	100.0
(N)	(145)	(218)	(363)	(208)	(190)	(265)

One-sided Fisher's exact test *P*-values for ≤9 *versus* ≥10 comparisons.

Place	*P*-value
North *versus* South Minneapolis inner-city	$0.53\ 10^{-4}$
Minneapolis inner-city *versus* outer-city	0.41
St Paul inner-city *versus* outer-city	$0.13\ 10^{-5}$

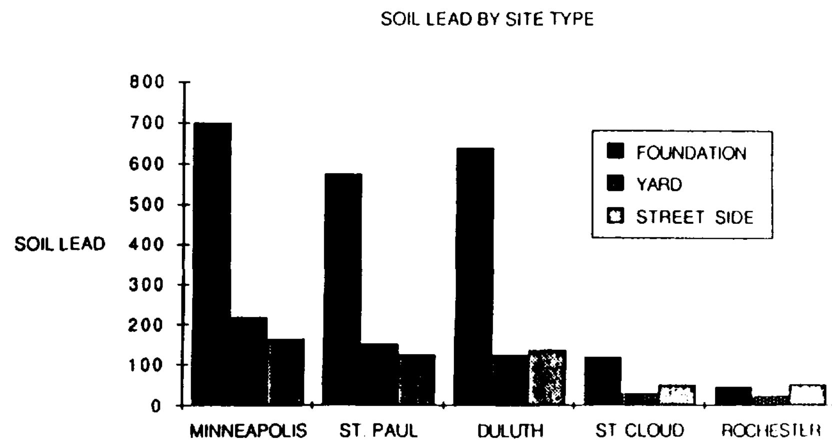

Figure 3. *Median soil lead level by collection type for each city sampled.*

rural background lead concentrations, the soils of major urban residential communities exhibit lead concentrations whereby 27%, 26%, 32% and 42% of the yards of Duluth, inner city North Minneapolis, inner city St Paul and inner city South Minneapolis, respectively, contain over 300 mg Pb/kg. In these same communities the childhood blood-lead measurement equal to or exceeding the current toxic guideline of 25 μg/dL is 0.7% in Duluth, 1.4% in inner city North Minneapolis, 6.3% in inner city St Paul and 6.4% in inner city South Minneapolis. All of these rates of childhoodlead exposure are unacceptable in modern society. The factors which have caused this situation require diligent attention.

Children as Biobindicators of Environmental Lead

Children are excellent biobindicators of available lead sources in the environment. This results from their developmental stage in which they crawl and play on the ground and floor and place their hands and playthings into their mouths. The exposure of children to lead has been directly related to the soil-dust lead concentrations found in the environment (Sayre *et al.*, 1974, 1981). Minnesota agency staff suggest elsewhere in this conference proceedings that there is a weak relationship between blood lead and soil lead. That conclusion is based on the small matched pairs data set described previously. The matched pairs data set is biased towards children with extremely high lead exposures as indicated by the fact that over 25% of the Minneapolis children reported in the matched pairs data set exhibited blood lead contents equal to or above 30 μg/dL. By contrast, 1.7% of Minneaplois children exhibited blood lead contents of 30 μg/dL or more in the unpaired data set. Comparing the two sets of data shows that they do not describe the same population (Fisher's exact test P-value $\leq 10^{-25}$). The large unpaired data set indicates that there is a strong association between blood lead and soil lead.

Numerous studies have demonstrated that when soils and dusts are contaminated with lead, children exposed to that soil and dust also have increases in their blood lead concentrations. The tendency for auto-exposure to lead by children has been demonstrated for a variety of particle sources such as street dust (Brunekreef *et al.*, 1981; 1983), house dust (Brunekreef *et al.*, 1981; Angle *et al.*, 1974), soil or dust (Brunekreef *et al.*, 1981, 1983; Angle *et al.*, 1974, 1975; Angle and McIntire, 1982; Cohen *et al.*, 1973; Reeves *et al.*, 1982; Shellshear *et al.*, 1975), or dust on workers' clothing (Baker *et al.*, 1977; Dolcourt *et al.*, 1978). The average child playing in ordinary ways consumes about 20 to 50 mg of soil per day, but the child exhibiting *pica* (consumption of non-food items) may easily consume 5 g or more of soil or dust each day (La Goy, 1987). Given that children absorb and retain about 50% of the lead they ingest, 50 mg of 100 μg/kg soil or dust would expose a child to 25 μg of lead per day from a single source. Ingestion of 1 to 5 g of soil would expose a child to between 50 and 250 μg of lead: 100 to 150 μg lead per day is the maximum allowable intake from all sources for the most sensitive children, from birth to two years of age (Mahaffey, 1977). Soil has therefore been understood to be a potent source of lead exposure and the likelihood of childhood exposure rises with increasing soil lead concentrations.

House Paint as a Factor in Population Lead Exposure

Conventional wisdom holds that house paint is the major source of lead contamination and childhood lead exposure. If true, then children living in cities and communities with aged housing should have r or less the

Table 9. *Housing construction dates by community.*[a]

Decade of construction	Minneapolis inner-city South	Minneapolis inner-city North	Minneapolis outer-city	St Paul inner-city	St Paul outer-city	Duluth	St Cloud	Rochester Old	Rochester New
1970–1979	14.8	14.9	3.1	9.6	10.1	13.5	41.9	4.2	50.4
1960–1969	19.9	12.2	8.2	8.3	13.2	7.5	20.1	6.6	28.7
1950–1959	6.5	5.9	7.4	6.8	14.6	12.2	12.1	15.0	9.8
Pre 1950	58.8	67.1	81.3	75.3	62.1	66.8	25.9	74.2	11.1
Blood lead (N)	(218)	(145)	(208)	(190)	(265)	(143)	(45)	(44)	(8)

Source: 1980 Census of Housing.
[a]This table includes only census tracts which were part of the blood lead survey.

same problem regardless of size of the community, and all old lead-painted dwellings should have the same soil lead concentration regardless of urban location.

The role of leaded paint in community contamination and lead exposure of populations of children can be assessed from the data set collected by the State. The age of a dwelling is often used as a proxy for the presence of leaded paints. Prior to the 1960s, house paints contained up to 30–50% lead by weight. Table 9 shows census tract data of the collective percentage of dwellings built during each decade for each community. The table includes only census tracts where children's blood lead concentrations were actually measured, and it emphasizes the fact that, in general, the oldest census tracts in the state of Minnesota were sampled. Note that outer city Minneapolis, "old" Rochester and inner city St Paul are the communities with the oldest dwellings. These are not the communities with the highest lead concentrations. In fact, "old" Rochester has some of the lowest lead concentrations in the state survey. Excluding St Cloud and "new" Rochester, the communities with the newest dwellings are inner city South Minneapolis, outer city St Paul and Duluth. The soils and children living in inner city South Minneapolis exhibit the highest lead concentrations in the state. There is no discernible general pattern between the age of dwellings within a community and the lead concentrations of either the soil or the blood of the childhood population of the same community.

Numerous multi-source studies have been done that included both house paint and soil or dust as a part of the evaluation (Angle *et al.*, 1974; Charney *et al.*, 1980; EHCLS, 1986; Guiguere *et al.*, 1977; Landrigan *et al.*, 1975; Rabinowitz *et al.*, 1984; Rice *et al.*, 1978; Sayre *et al.*, 1974, 1987; Watson *et al.*, 1978; Yankel *et al.*, 1977). None of these population studies found a significant relationship between blood lead and leaded paint, but this same group of studies did find a positive relationship between blood lead concentrations and either soil or dust. Further evidence concerning leaded paint was gathered in a study of children with high blood lead concentrations to determine whether they had paint chips in their stools. Hammond *et al.*, (1980) examined faecal samples of highly exposed children who lived in homes with leaded paint. He expected to find paint chips and intermittent high doses in the stool. But in many children he found relatively high lead concentrations evenly mixed throughout the stool without paint chips or lead spikes. In a follow up study he concluded that lead exposure was due to lead dusts and that it could not be established whether or not lead paint was the source (Hammond, 1982). The US Environmental Protection Agency noted that based upon published and unpublished Centers for Disease Control data, a source of lead paint hazard could not be confirmed for about 40 to 50% of the cases of children with elevated blood lead concentrations (EPA 1986, 11–160).

The literature, as a whole, supports what was found in Minnesota. The mere presence of leaded paint does not explain population blood lead concentrations. Angle *et al.*, (1975) examined blood lead concentrations from the perspective of the geographic location of dilapidated housing, high traffic roads and point sources of lead in a city. The distribution of blood lead concentrations matched the location of point sour[illegible] of lead and

traffic, but not dilapidated housing. The presence of flaking, peeling paint is insufficient, by itself, to raise blood lead concentrations of a population of children. Yet, for an individual child, house paint may be an extremely important source of lead. This is especially true when the paint has been pulverized (by sanding and burning) into small particles. Recent studies show that lead paint abatement and redecorating of dwellings significantly raises the risk of lead exposure in children compared to their previous condition (Amitai *et al.*, 1987; Chisolm *et al.*, 1985; and Sayre, 1987).

Lead Quantities as a Function of City Size and Community Traffic Flows

If age and presence of house paint cannot account for the urban lead pattern, then what is the missing lead source that varies with city size? Lead, as an additive to petrol, has been exhausted to the atmosphere in large quantities. The amount of lead exhausted by vehicles varies with traffic flow, which in turn is determined by the population of drivers and distance driven within a given city. In this way, the size of city plays a role in the quantity of lead that has accumulated within a given place. In large cities distances are grater, journeys are longer and, because of sheer volume of vehicles, traffic congestion and the amount of petrol consumed is greater. In small cities, distances are smaller, journeys are shorter, and the smaller number of vehicles reduces traffic congestion and the amount of petrol consumed.

Tables 1–8 show the soil lead and blood lead of children by city and community subset. There is a consistent trend of soil or dust lead and blood lead that corresponds with city size. The largest cities have the highest lead concentrations and the smallest cities have the lowest lead concentrations. Traffic congestion and the use of leaded petrol over the decades has had the effect of causing a disproportionate contamination of large cities compared with small cities. In this way, soil or dust lead accumulation has been a function of city size, which in turn has had an impact on childhood exposure to lead. These ideas are supported by a microanalytical technique for the physiochemical characterization of lead in urban dusts which found that lead from petrol contributes substantially to dusts collected around buildings having lead-painted trim and situated some distance from a roadway (Linton *et al.*, 1980). EPA (1986) concluded that soil lead concentrations near houses were due to both exterior leaded paint and leaded petrol.

Within cities, traffic flow and, hence, the accumulation of lead, is not equally distributed. Because of their locations, some communities have had high traffic flows for years and these places show up with comparatively high lead accumulation and exposure. For example, inner city Communities often have fundamentally higher soil or dust and blood lead concentrations than outer city areas. Traffic flow converges toward the inner city in a manner which disproportionately increases the inner city lead accumulation compared with equally old, but outer city urban communities. Tables 5–8 show the soil lead and blood lead concentrations foun[illegible]nner city compared with outer city in St Paul and Minneapolis. St

Paul has major differences in soil lead and childhood exposure to lead between inner and outer city locations. Although Minneapolis shows a relatively strong difference in environmental concentrations between inner and outer city locations, childhood exposure to lead is not significantly different for this particular stratification of the city. For Minneapolis, the role of traffic flow in the urban accumulation of lead is most strongly demonstrated by comparing the environment and exposure concentrations between the inner city communities of North and South Minneapolis.

As shown in Tables 5–7, inner city South Minneapolis has the highest soil lead concentrations. Note that the housing of South Minneapolis is somewhat newer than the housing of North Minneapolis. The major difference between these two communities is the history of traffic flow within each community. North Minneapolis has been the site of local traffic rather than through traffic. In contrast, South Minneapolis has been a site of greater commercial activity and has long been riddled with major roadways that carry heavy traffic volume between St Paul and Minneapolis, to the southern and western suburbs of Minneapolis as well as between Minneapolis and points south and west of the Twin Cities. The difference of exposure to lead between inner city North and South Minneapolis strongly supports the idea that traffic- derived lead aerosols have been a potent substance in environmental contamination and lead exposure of the general population of children.

The findings from Minnesota communities are supported by numerous other studies which compared soil and blood lead concentrations between large and small communities of implicitly different traffic flows. Studies of Los Angeles, California and the nearby community of Lancaster (Johnson *et al.*, 1975) ; Rotterdam and the Hague, The Netherlands and their outlying communities (Brunekreef *et al.*, 1983); Hartford, Connecticut and a nearby suburb (Cohen *et al.*, 1973); and Omaha, Nebraska and suburb (Angle *et al.*, 1974) all showed the same patterns as described above for Minnesota. Many additional studies have been done that report only blood lead concentrations and support the findings in Minnesota cities. The best of these studies was the NHANES II data, which showed that for all age groups, races and socio-economic classes, people living in urban areas with a population of one million or more had higher blood lead concentrations than urban areas of less than one million, which in turn had higher blood lead concentrations than rural areas (NCHS, 1984). Also, when large cities were examined, blood lead concentrations of inner cities were found to be higher than outer cities (NCHS, 1984). The NHANES II data demonstrate that as lead aerosols from petrol are reduced, the blood lead concentrations of the population were also decreased; but the urban child most exposed to lead experienced smaller reductions in blood lead concentration than the suburban child least exposed to lead (Annest *et al.*, 1983). Higher blood lead concentrations have also been reported in Philadelphia, Chicago and New York compared with corresponding suburban areas (Hasselblad and Nelson, 1975). Dublin children have higher blood lead concentrations than children of surrounding areas (Richardson *et al.*, 1982). In Sweden, Trelleborg children have higher blood lead concentrations than rural Scania children (Skerfving *et al.*, 1986). Young people of T vo had higher

blood lead concentrations than suburban young people (Tsuchiya *et al.*, 1977; Okubo *et al.*, 1978).

The relative contribution of leaded petrol and leaded paint to urban soils has also been studied. EPA (1986) found that the emissions from leaded petrol are predominately small particles (6–8 μm). Given that most urban areas do not have lead industries and that air lead is predominately small particles (>80%), leaded petrol is the principal constituent of air lead (EPA, 1986). Attempts to calculate a more specific determination of sources of lead in soil, dust and house dust have been undertaken. Fergusson and Schroeder (1985) concluded that leaded petrol accounted for 90% of the house-dust lead in new developments and 50% of the house-dust lead in "older" developments with 45% of the remainder due to leaded paint. Since about half of all house dust is soil dust and 87% of street dust is from soil lead (Fergusson, 1986), and soil lead is predominantly due to air lead (EPA, 1986), and air lead is predominately leaded petrol, it is reasonable to conclude that soil lead, street-dust lead, and much of house-dust lead is primarily from leaded petrol. This idea is supported by Sturges and Harrison (1985) who found that soil lead was less than 20% leaded paint and that house dust was less than 15% leaded paint. As a group, these studies emphasize the role that the accumulation of small aerosol particles play in exposing children to lead. Whenever there is lead dust generated and deposited into a child-accessible environment, the risk and probability of childhood lead exposure increases. Because of traffic flow patterns and the legacy of the consumption of leaded petrol, the largest and most densely populated urban areas of Minneapolis, St Paul and Duluth have become the highest risk environments in Minnesota. It is very likely that the same patterns will be found in every major city of the nation.

Conclusions: The Case for Lead Contaminated Soil Abatement in Minnesota

The case for abatement of lead contaminated soil or dust in Minnesota can be made on the basis of the survey results and information from the literature. There is good definition as to which geographical areas have the highest risk of lead exposure. Children living in the most traffic-congested communities of the three largest cities in Minnesota (Minneapolis, St Paul, and Duluth) exhibit the highest rates of lead toxicity. There is no indication that children living in smaller cities are being similarly exposed to lead. Furthermore, the margin of safety from lead exposure to children living in the largest cities is non existent or extremely narrow, whereas the margin of safety from lead exposure to children living in the smaller cities appears to be excellent.

At the rates of exposure demonstrated by the blood lead survey, Minneapolis, St Paul, and Duluth have nearly 1,500 children with blood lead concentrations 25 μg/dL and above. Another 6,500 children have blood lead concentrations from 15 to 25 μg/dL. Considering the numbers of children being excessively exposed to lead, the health and social costs of doing r 'ing are extremely high. It has been estimated that as a result of lead exposure in Minnesota, the direct costs of medical and compensatory education and indirect costs due to reduced future earnings amount to between $15 and $17 million per year (Reagan, 1988).

Leaded paint alone does not appear to be the major problem in the accumulation of soil lead or the lead exposure of childhood populations. The fact that foundation soils contain high lead concentrations is most likely due to impaction of lead aerosols on building sides and the wash-off of these particles into the soils near the foundations of buildings. When paint chips are present they spike soils with high doses of lead. There is evidence that without extreme caution, emphasis on cleanup of leaded paint would exacerbate rather than reduce lead exposure to childhood populations. In addition to the hazard it creates, paint removal is very costly, whereas repainting and thorough dust cleanup is relatively inexpensive.

The major patterns of soil lead in Minnesota are related to city size and traffic flow. The accumulation of extremely small aerosol particles from leaded petrol is directly linked to environmental contamination and lead exposure of childhood populations in Minnesota. Given that the Minnesota data were collected after the rapid phase-down of leaded petrol, there is no indication that further substantial reductions in lead exposure are possible without abatement of the residual lead in soil-dust that has accumulated in the most populated areas of the major cities. Improvements in the quality of urban residential environments are necessary in order to broaden the margin of safety to prevent lead toxicity for the most exposed populations of children in Minnesota. Cleanup of lead contaminated soil or dust can be done as part of a relandscaping and dust control programme and should be relatively inexpensive and effective at reducing population lead exposure.

A critical question is the safe lead concentration in soil that protects children from undue exposure. Research on dose-response curves, as well as related studies, show that a rapid rise in population blood lead concentrations takes place when the lead content of soil increases from less than 100 mg/kg to 500–600 mg/kg and then the curve flattens off (Angel and McIntyre, 1982; Bornschein *et al.*, 1986; Brunekreef *et al.*, 1981; Chaney and Mielke, 1986; Reeves *et al.*, 1982; and Vimpani, 1985). Shellshear *et al.*, (1975) evaluated children in Christchurch, New Zealand, and concluded that children exposed to more than 100 mg Pb/kg lead in soil and who also exhibit pica are at major risk to lead exposure. The empirical data from Minnesota provide further support for these previous findings. Children living in the small cities of St Cloud and Rochester do not exhibit excessive lead exposure and provide a basis for developing a cleanup standard. In these cities, 40% of the streetside and 70% of yard soils contain lead amounts below 50 mg/kg, and only a small percentage of soils are above 150 mg/kg. In the three largest cities, when 40% of the soils exceed 150 mg/kg, then the population of children no longer appears to have a sufficient margin of safety to prevent a portion of the children from exhibiting lead toxicity.

In sum, Minnesota has a good base of information that includes the rate of childhood lead exposure, the source and geography of environmental

contamination, and the environmental lead concentrations that are protective to populations of children. This information provides the necessary scientific basis for developing a primary lead prevention programme to reduce the lead concentrations of the most exposed children in Minnesota.

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References

Amitai, Y., Graef, J., Brown, M., Gerstle, R., Kahn, N. and Cochrane. P. 1987. Hazards of "deleading" homes of children with lead poisoning, *AJDC*, **141**, 758–760.

Angle, C., McIntire, M. and Colucci, A. 1974. Lead in air, dustfall, soil, housedust, milk, and water: correlation of blood lead in urban and suburban school children. *Trace Substs Envir. Health*, **8**, 23–29.

Angle, C., McIntire, M. and Vest, G. 1975. Blood lead of Omaha school children — topographical correlation with industry, traffic and housing. *Neb. Med. J.*, **60**, 97–102.

Angle, C. and McIntire, M. 1982 Children, the barometer of environmental lead. *Adv. Pediat.*, **29**, 3–31.

Annest, J., Pirkle, J., Makuc, D., Neese, J., Bayse, D. and Kovar, M. 1983. Chronological trend in blood lead levels between 1976 and 1980. *New England J. Med.*, **308**, 1373–1377.

Baker, E., Folland, D., Taylor, T., Frank. M., Peterson, W., Lovejoy, G., Cox, D., Housworth, J. and Landrigan, P. 1977. Lead poisoning in children of lead workers: home contamination with industrial dust. *New England J. Med.*, **296**, 260–261.

Bornschein, R., Succop, P., Krafft, K., Clark, C., Peace, B. and Hammond, P. 1986. Soil lead, interior house dust lead and childhood lead exposure in an urban environment. *Trace Substs. Envir. Health*, **20**, 322–332.

Brunekreef, B., Veenstra, S., Biersteker, K. and Boleij, J. 1981. The Arnhem lead study: I Lead uptake by 1b3 year old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. *Envir. Res.*, **25**, 441–448.

Brunekreef, B., Noy, D., Biersteker, K. and Boleij, J. 1983. Blood lead levels of Dutch children and their relationship to lead in the environment. *J. Air Poll. Cont. Assoc.*, **33**, 872–876.

Charney, E., Sayre, J. and Coulter, M. 1980. Increased lead absorption in inner city children where does the lead come from? *Pediatrics*, **65**, 226–231.

Chisolm, J., Mellits, E. and Quaskey, S. 1985. The relationship between level of lead absorption in children and the age, type and condition of housing. *Envir. Res.*, **38**, 31–45

Chisolm, J., Mellits, E. and Quaskey, S. 1985. The relationship between level of lead absorption in children and the age, type and condition of housing. *Envir. Res.*, **38**, 31-45

Cohen, C., Bowers, G. and Lepow, M. 1973. Epidemiology of lead poisoning: a comparison between urban and rural children. *JAMA*, **226**, 1430–1433.

Dolcourt, J., Hamrick, H., O'Tuama, L., Wooten, J. and Barker. E. 1978. Increased lead burden in children of battery workers: asymptomatic exposure resulting from contaminated work clothing. *Pediatrics*, **62**, 563–566.

EHCLS. 1986. *East Helena Child Lead Study*. East Helena Montana Department of Health and Environmental Sciences, Lewis and Clark County Health Department. Centers for Disease Control, Atlanta, Georgia.

EPA. 1986. *Air Quality Criteria for Lead*. Environmental Criteria and Assessment Office. Rese Triangle Park, N.C.

Fergusson, J. and Schroeder, R. 1985. Lead in housedust of Christchurch, New Zealand: sampling, levels and sources. *Sci. Total Envir.*, **46**, 61–72.
Fergusson, J. 1986. Lead: petrol lead in the environment and its contribution to human blood lead levels. *Sci. Total Envir.*, **50**, 1–54.
Giguere, C., Howes, A., McBean, M., Watson, W. and Witherell, L. 1977. In-creased lead absorption in children of lead workers, Vermont. *MMWR*, **26**, 61–62.
Hammond, P., Clark, C., Gartside, P., Berger, O., Walker, A. and Michael, L. 1980. Fecal lead excretion in young children as related to sources of lead in their environments. *Intl. Arch. Occ. Envir. Health*, **46**, 191–202.
Hammond, P. 1982. Exposure to lead. In: J. Chisolm *et al.* (eds.), Lead Absorption in Children, pp. 55–61. Urban and Schwarzenberg, Baltimore.
Hasselblad, V. and Nelson, D. 1975. Additional analysis of the seven city lead study, In: T. Griffin and J. Knelson (eds.), *Lead*, pp. 147–151. Academic Press, New York.
Johnson, D., Tillery, J. and Prevost, R. 1975. Levels of platinum, palladium, and lead in populations of Southern California. *Environ. Health Perspect.*, **12**, 27–33.
La Goy, P. 1987. Estimated soil ingestion rates for use in risk assessment. *Risk Analysis*, **7**, 355–359.
Landrigan, P., Gehlbach, S., Rosenblum, B., Shoults, J., Candelaria, R., Banthel, W., Liddle, J., Smrek, A., Staehling, N. and Sanders, J. 1975. Epidemic lead absorption near an ore smelter: the role of particulate lead. *New England J. Med.*, **192**, 123–129.
Linton, R., Natusch, D., Solomon, R. and Evans, C., Jr. 1980. Physiochemical characterization of led in urban dusts: a microanalytical approach to lead tracing. *Environ. Sci. Tech.*, **14**, 154–164.
Mahaffey, K. 1977. Relation between quantities of lead ingested and health effects of led in humans. *Pediatrics*, **59**, 448–456.
Martin J. and Lanegran, D. 1982. *Where We Live. The Residential Districts of Minneapolis and Saint Paul*, University of Minnesota Press, Minneapolis in association with the Center for Urban and Regional Affairs, University of Minnesota, Minneapolis.
Mielke, H., Anderson, J., Berry, K. K., Mielke, P., Jr., Chaney, R. and Leech, M. 1983. Lead concentrations in inner-city soils as a factor in the child lead problem. *Am J. Public Health*, 73(12), 1366–1369.
Mielke, H., Blake, B., Burroughs, S. and Hassinger, N. 1984. Urban lead levels in Minneapolis: the case of the Hmong children. *Environ. Res.*, **34**, 64–76.
Mielke, H., Burrough, S., Wade, r., Yarrow, T. and Mielke, P., Jr. 1984/85. Urban lead in Minnesota: soil transect results of four cities. *Minnesota Acad. Sci.*, **50**(1), 19–24.
MCHS. 1984. Blood lead levels for persons 6 months to 74 Years. *Vital and Health Statistics*, **11**(233), pp. 29–31. National Center for Health Statistics.
Okubo, T., Tsuchiya, K., Nagasaki, M., Nakajima, T., Kamijyo, H. and Mizoguchi, I. 1978. A further study of the biological effects of lead on urban and suburban Tokyo school children. *Intl. Arch. Occ. Environ. Health*, **41**, 17–23.
Pierce, F., Dowdy, R. and Grigal, D. 1982. Concentrations of six trace metals in some major Minnesota soil series. *J. Environ. Quality*, **11**(3), 416–422.
Rabinowitz, M., Needleman, H., Burley, M., Finch, H. and Rees, J. 1984. Lead in umbilical blood, indoor tap water and petrol in Boston. *Arch. Environ. Health*, **39**, 299–301.
Reagan, P. 1098. The social costs of childhood lead poisoning. Unpublished report written for The Lead Coalition, 1051 S. McKnight Road, St Paul, MN 55119.
Reeves, R., Kjellstrom, T., Dallow, M. and Mullins, P. 1982. Analysis of lead in blood, paint, soil and housedust for the assessment of human lead exposure in Auckland. *New Zealand J. Sci.*, **25**, 221–227.
Rice, C., Fischbein, A., Lilis, R., Sarkozi, L., Kon, S. and Selikoff, I. 1978. Lead contamination in the homes of employees of secondary lead smelters. *Environ. Res.*, **15**, 375–380.
Richardson, R., Jeffrey, D. and Kevany, J. 1982. Lead in petrol (Letter). *Brit. Med. J.*, **284**, 1705–1706.
Sayre, J., Charney, E., Vostal, J. and Pless, I. 1974. House and hand dust as a potential source of childhood lead exposure. *AJDC*, **127**, 167–170.
Sayre, J. 1981. Dust lead contribution to lead in children. In: D. Lyman *et al.* (eds.), *Environmental Lead*, pp. 230–40. Academic Press, New York.
Sayre, J. 1987. Deleading houses: danger in the dust. *AJDC*, **141**, 727–728

Shellshear, I., Jordon, L., Hogan, D. and Shannon, F. 1975. Environ-mental lead exposure in Christchurch children: soil lead as a potential hazard. *New Zealand Med. J.*, **81**, 382–386.
Skerfving, S., Schutz, A. and Ranstam, J. 1986. Decreasing lead exposure in Swedish children, 1979–1984. *Sci. Total Environ.*, **58**, 225–229.
Sturges, W. and Harrison, R. 1985. An assessment of the contribution from plain flakes to the lead content of some street and household dusts. *Sci. Total Environ.*, **44**, 225–234.
Tsuchiya, K., Okubo, T., Nagasaki, M., Nakajima, T., Kamijyo, H. and Mizoguchi, I. 1977. Biologic effects of lead on school children of urban and suburban Tokyo. *Intl. Arch. Occ. Environ. Health*, **38**, 247–257.
Vimpani, G. 1985. The Port Pirie Cohort Study: blood lead concentration and childhood development assessment In: L. Goldwater *et al.*, (eds.), *Lead Environmental Health. The Current Issues*, pp. 139–146. Duke University, Durham, N.C.
Watson, W., Witherell, L., Giguere, G. 1978. Increased lead absorption in children of workers in a lead storage battery plant, *JOM*, **20**, 759–761.
Yankel, A., Lindern, I. and Walter, S. 1977. The Silver Valley lead study: the relationship between childhood lead levels and environmental exposure. *J. Air Pollut. Contam. Assoc.*, **27**, 763–767.